THE ETIOLOGY OF EMPYEMA IN CHILDREN:

AN EXPERIMENTAL AND CLINICAL STUDY.

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(Concluded from p 50.)

EXPERIMENTS UPON ANIMALS.

Rabbits, diplococcus pneumoniæ.

1. Medium-sized, white rabbit, injected in pleural cavity, with a pure culture in bouillon of the diplococcus pneumoniæ; died in eighteen hours.

Autopsy.—Rigor mortis marked; chest on both sides showed pleurisy of a sero-fibrinous character; fibrin of a greenish-yellow tint; serum more abundant in left chest. Pericardium: pericarditis, with slight amount of fibrin. Peritoneum: a slightly increased serum; no fibrin; no evidence of inflammation. Blood gave upon culture and stain diplococcus pneumoniæ (pure). Pleuritic fluid, diplococcus pneumoniæ pure culture.

2. Black rabbit (small). Inoculated March 17th; killed by medulla while dying, March 21st. Injected pure culture diplococcus pneumoniæ in chest.

Autopsy.—Sero-fibrinous pleurisy, double sero-fibrinous pericarditis. Peritonitis, sero-fibrinous exudate. Spleen large and swollen. Blood and pericardial fluid pure culture diplococcus pneumoniæ.

3. Small white rabbit; injected about ten minims bouillon culture in chest of diplococcus, March 18th.

Autopsy, March 22.—Beginning rigor mortis. Slight pleuritis; very little serum and fibrin in pleuritic cavity. Sero-fibrinous exudate in peritoneal cavity (peritonitis). Spleen not very large. Blood gave pure culture. diplococcus in agar and bouillon, no growth in gelatin, and found also by stain. Lung juice crude cover-glass gave exquisite capsule diplococci.

4. Rabbit, medium sized, injected with pure bouillon culture diplococcus pneumoniæ; died instantly; failure of injection.

5. Rabbit, medium sized; injected in chest same as 4; died on the fourth day.

Autopsy.—Pleuritis double sero-fibrinous; very severe. Pericarditis sero-fibrinous, pericarditis marked. Spleen large and soft. No peritonitis; no meningitis. Specimens of blood, pleuritic and pericardial fluid inoculated on agar, gelatin, bouillon, etc.; reaction by stain and culture of diplococccus pneumoniæ.

6. Rabbit, medium size, injected in chest with pure culture of diplococcus pneumoniæ; died in six days.

Autopsy.—Double sero-fibrinous pleurisy. Pericarditis sero-fibrinous. Peritonitis sero-fibrinous. Spleen large. Cultures made from blood and pericardial fluid; diplococcus pneumoniæ.

7. Rabbit, medium size, injected subcutaneously with pure culture of diplococcus pneumoniæ; no result.

8. Rabbit, black and white spotted, medium size; injected in chest with five minims of empyema pus in which diplococci pneumoniæ existed; animal died in thirty-six hours.

Autopsy.—Forty-eight hours after injection. Rigor mortis marked. Pleurisy slight; some fibrin in the left side at point where needle entered the lung. Peritonitis very marked with sero-fibrinous exudate; brownish serum in bottom peritoneal cavity. Cultures from peritoneum and pleura, pure culture diplococcus pneumoniæ.

9. Small, active rabbit, injected with pure culture diplococcus pneumoniæ obtained from animal 8.

Autopsy made before rigor mortis. Pleurisy marked; sero-fibrinous exudate on both sides. Pericarditis with adhesions; sero-fibrinous exudate. Spleen large. Peritonitis marked; sero-fibrinous exudate. Pure cultures of diplococcus pneumoniæ obtained from pleura, pericardium and blood.

10. Rabbit, medium-sized animal, injected with peritoneal fluid of animal 9; died in two days.

Autopsy.—Four hours post-mortem. Pleurisy left sero-fibrinous. Pericarditis slight. Peritonitis marked. Spleen enlarged. Cultures show diplococcus pneumoniæ.

11. Rabbit, large white doe, injected with twenty minims of peritoneal fluid of rabbit 10, in each lung ten minims; killed by medulla on seventh day in dying condition.

Autopsy.—Marked sero-fibrinous pleurisy on both sides and also pericarditis. Spleen large.

12. Rabbit, small-sized, lively animal; injected with peritoneal fluid of rabbit 8; first two days considerable dyspnœa, but recovered and was apparently well. Killed after a week; the only thing found were a few delicate adhesions between the coils of intestine; some clear fluid in bottom of peritoneal cavity.

13. Rabbit, medium-sized; pure culture injection; killed after fifth day.

Autopsy showed marked sero-fibrinous pleurisy; marked sero-fibrinous pericarditis, also advanced peritonitis, with sero-fibrinous exudate. Large spleen. Pure culture of diplococci obtained from blood, pleuritic and pericardial fluid.

14. Rabbit; died with a paralysis of posterior extremities; exhaustion; remains of an old pleurisy found; adhesions of the costal and pulmonary pleuræ. This animal was injected in the pleura with pure culture streptococcus pyogenes.

15. Rabbit; injected with pure culture streptococcus pyogenes in pleural cavity from Case XI.; died after two days.

Autopsy.—Kidneys larger than normal and congested, but the spleen was enormous in size. Cultures of blood gave streptococci.

16. Rabbit; subcutaneous injection streptococcus pyogenes (pure); death after three days.

Autopsy.—Jaundice, universal, of all tissues. Lungs contained small abscess in lower right lobe. Heart, normal. Liver, very small abscesses, studded throughout. Kidneys, cloudy swelling. Spleen much enlarged. Urine, bile pigment.

17. Rabbit; injected with pure culture streptococcus pyogenes, with no result; lived for months.

In addition to the above experiments, there were some injections into guinea-pigs of the diplococcus pneumoniæ; these resulted negatively, except one animal which had been injected with pleuritic fluid from a rabbit that had died in its turn from an injection of diplococcus. In this there were typical pleurisy, pericarditis, and peritonitis, with large spleen.

A few injections upon rats and mice subcutaneously resulted in case of diplococcus positively in one case. Injections into additional rabbits with pus obtained from a tubercular case gave nothing worthy of note.

CASES.

CASE I.—Y. S., aged seven years, admitted into hospital. Mother has had phthisis; child has had measles, four years ago, since then a slight cough. Became sick suddenly two weeks ago; has been ill since with cough, pain in the side, chilly sensations, dyspnœa.

Physical signs (left side). Flatness from mid-scapula down. Bronchial voice and breathing (at middle scapula), below distant breathing. Signs of bronchitis on both sides; no signs of phthisis in front or on opposite side.

Operation eighth rib, resected in the post-axillary line; abscess was found to be higher up and encapsulated; adhesions had to be broken down in order to reach empyema. Examination of pus: crude cover and cultivation; streptococcus pyogenes. Cured.

CASE II.—R. T., male, aged eight years; January 1st. Family history negative. He had scarlet fever four years ago; a year ago had a bronchitis; a month ago also bronchitis (?). Two weeks ago had fever, temperature 104° (physician). Complained of headache; next day had pain in the left side; dyspnœa; slight cough; no expectoration; temperature 103°. During the next week the temperature did not exceed 101.5°.

Admission to hospital, showed marked œdema of skin of left side, but on incision later (at operation) no infiltration of pus was seen; signs of fluid in left side.

January 2. Operation, resection as noted; there was no infiltration of the œdematous skin with pus. In spite of resection the lung would not expand.

January 3 *to February* 10. Temperature 100°, 101°, to 104°; pulse 110, 112, 138, to 152; respiration 32, 44, to 52. The boy did badly; there was a profuse discharge of pus, fetid in odor at each dressing, and lung did not expand. Several resections were performed upon this chest to favor retraction of chest toward lung and closure of the large cavity in the chest. There resulted much deformity, and at discharge from hospital there existed a discharging fistula.

Bacterioscopic results were limited to stainings of cover-glass specimens of pus and showed tubercle bacilli. Animal experiments negative.

CASE III.—I. S., aged two years; male; February 18, 1890. Has had measles, a year ago; inflammation (?) of the lungs ten months ago; present illness began with fever six weeks ago; this fever after a few days was complicated by convulsions recurring at intervals. Cough was very marked and present during the entire illness; there was loss of flesh and strength, and increasing pallor.

Admission to hospital showed a weakly child, badly nourished, pale; temperature 100°; respirations 40; pulse 120. Physical signs of fluid in right side chest. Tenth rib exsected; about a pint of thick, green, tenacious pus evacuated.

Bacterioscopic result of examination, pus; diplococcus pneumoniæ (Fränkel and Weichselbaum).

May 11. Discharged cured.

CASE IV.—I. S., male, aged eleven months; has been sick for three weeks with fever, cough, dyspnœa, emaciation. Signs of fluid in right pleural cavity. Dulness to flatness. Loss of movement. Loss of fremitus. Loss of voice; pleuritic râles over whole side; syringeful yellow, thick pus removed.

Bacterioscopic examination. Staphylococcus pyogenes aureus. Result unknown.

CASE V.—P. M., male, aged two and a half years; March 3, 1890. Previous history negative. Present illness began a month ago with fever, cough, difficulty in breathing, and constitutional disturbances; fever continued marked during the first two weeks, then abated, but continued nevertheless; pallor, loss of flesh and strength, and generally becoming worse.

Admission to hospital. Anæmic, poorly nourished, anorexia, cough. prostration, and fever; temperature 101°; respirations 46; pulse 104, Signs of fluid in the left side and areas of consolidation (broncho-pneumonia) over the right side. Heart apex just inside the middle line.

March 4. Turbid flocculent serum removed from the chest. Cultures show diplococcus pneumoniæ. Aspiration, only four fluidounces (serous) removed in all, but patient continued to grow worse, temperature varying from 101° to 104°; respirations from 50 to 70; pulse 148 to 160.

16*th*. Needle in left side withdrew a creamy pus.

18*th*. Resection of rib, and about one ounce of pus evacuated, but patient grew worse; died March 27th from broncho-pneumonia on both sides.

Examination (bacterioscopic) of pus withdrawn March 16th, showed diplococcus pneumoniæ.

CASE VI.—I. K., male, aged three and a half years; March 13, 1890. Family and personal history not lucid. Present illness began nine weeks ago with fever and cough and difficulty in breathing; prostration, loss of appetite; patient complained of pain in the left side; loss of flesh and strength; there were night-sweats; patient has steadily lost ground.

Admission to hospital. Poorly nourished, anæmic, delicately built lad; no sign of rhachitis; temperature 101°; respirations 40; pulse 132 (dyspnœa). Physical signs showed fluid in left side. Heart apex pushed to the right, and sounds most intense behind lower end of sternum; no signs of phthisis at apices.

March 16. Operated resection of eighth rib; greenish odorless pus, with large masses of fibrin evacuated, as big as a child's hand.

28*th*. Discharged cured.

Bacterioscopic examination of pus, diplococcus pneumoniæ.

CASE VII.—M. B., aged two years; male; March 1, 1890. Family and personal history negative. The previous history is indefinite; the patient for some time has suffered from cough. Present illness dates a week back; contracted a severe cough, and dyspnœa became very marked; fever, night-sweats, loss of appetite.

On admission, patient is well nourished; suffers great dyspnœa, this symptom so marked as to give impression that œdema of the glottis was present, or obstruction in the larynx. Intubation attempted but failed, and tracheotomy performed. Temperature 100°. Examination of the chest revealed spots of broncho-pneumonia over both sides. March 5th to 15th, temperature 101° to 105°; respiration 40 to 76; pulse 140, with dyspnœa; increasing; attempts to remove tracheal tube resulted in renewed attacks of dyspnœa. March 15th, signs of fluid in left side of chest, and on March 16th, operated, and twelve ounces of pus, greenish, no bad odor, removed from left side. Eighth rib resected.

March 18. Discharged cured.

Bacterioscopic study and culture of pus revealed diplococcus pneumoniæ.

Case VIII.—H. G., male, aged twelve years. Father, mother, and sisters and brothers in good health; previous history indefinite. It is not possible to obtain definite information as to the exact time of onset of present illness. He has for a few weeks past suffered from cough, fever, and night-sweats, pain in the right side upon coughing or taking deep inspiration; has been confined to his bed; no history of traumatism.

Admission to hospital. An anæmic but well-built lad, not at all emaciated. Physical examination revealed fluid, and needle withdrew pus in right side. Temperature 100°; respiration 40; pulse 140; resection of rib on right side and a pint of greenish fluid, not adhesive pus, removed; no fibrin clots were withdrawn; some adhesions between the costal and pulmonary pleuræ.

Bacterioscopic examination of pus revealed streptococcus pyogenes.

Result: Cure.

Case IX.—Female, aged two and one fourth years. April 10, 1890. Family history shows nothing. Present illness began three weeks ago with some eruption (?) upon the body; about a week later there appeared a cough, fever, distress in stomach and abdomen.

Admission to hospital. Anæmic child, with signs of rhachitis, cough, dyspnœa, and some cyanosis; temperature 101°; pulse 160; respirations 48. Physical signs of fluid in the right side, pus withdrawn, immediate operation, exsection; sixteen ounces of pus evacuated. April 12th to 22d, temperature varied from 99° to 103°; respirations 40 to 43; pulse 130 to 158; on April 22d, symptoms pointing to an invasion of the healthy lung by broncho-pneumonia appeared, and patient died with all the symptoms of pneumonia and exhaustion.

Bacterioscopic examination of pus revealed diplococcus pneumoniæ (Fränkel and Weichselbaum).

Case X.—M. G., male, aged four years; April 18th. Family and previous history negative. Present illness began seven weeks ago with scarlet fever; three weeks later the patient developed general anasarca, this lasted until eight days ago; at the same time he had a severe cough, chills, and fever; constant sweating, lasting until the date of admission; loss of flesh and strength, also failure of appetite and frequent micturition.

Admission to hospital. Patient anæmic and emaciated, cough and dyspnœa. Temperature 99°; respirations 42, and pulse 140. Signs of fluid in the left side. Heart apex pushed to the right of the sternum.

Operation, exsection of rib; twenty-nine and a half ounces of pus of creamy character evacuated.

Bacterioscopic examination revealed diplococcus pneumoniæ.

Result: recovery.

Case XI.—Male child, aged four months. May 9, 1890. Had been perfectly well until two weeks ago, at this time child was vaccinated; simultaneously with the vaccination, there appeared a burrowing abscess on the dorsum of the left foot which was deep and passed between the metatarsal bones. Eight days ago a febrile movement appeared and the patient was referred by me to a surgeon for treatment. There were at this time no lung symptoms. Three days ago the child developed cough and dyspnœa.

Status præsens: There is cough, dyspnœa, sighing respiration; pulse increased in rapidity; temperature 103°; abscess on foot still present; vaccine pustule still size of five cent piece and angry-looking. Examination of left side revealed chest full of fluid and needle withdrew pus, light yellow, thin, easily separating into two layers, clear and serous, upper and lower purulent.

May 10. Exsection of rib; six ounces of pus (milky) evacuated.

12th. Died; no autopsy.

Bacterioscopic examination of pus revealed streptococcus pyogenes.

Case XII.—*May*, 1890. Male, aged twelve months; well developed; has been suffering from pertussis and just recovered; about two weeks before date was doing well, when suddenly there appeared a new cough with dyspnœa, and broncho-pneumonia of the right lung was diagnosticated. The cough and fever and dyspnœa still continued up to May 9th, when fluid was found in the right side. A needle introduced about this time withdrew a clear serum; temperature 103°; pulse 160, and rapid respiration. Transferred to the hospital, where child next day showed a measles eruption.

13th. Needle introduced into right side and pus was withdrawn; child was doing well, but on the morning of the above date, patient suddenly developed tympanites (peritonitis (?)) and died.

Bacterioscopic examination of pus of May 13th and serum of May 9th revealed diplococcus pneumoniæ.

Case XIII.—Empyema; perforation; spontaneous recovery. Female, aged thirteen months. May 12, 1890. Previous history negative. Child was taken ill about three weeks ago with cough, fever, dyspnœa. The child had dyspnœa, and moaned when it breathed; there was a frequent cough and a temperature of 103½°; pulse 160. Examination of chest showed consolidation of the left upper lobe (dulness, bronchial voice, and breathing). Diagnosis: broncho-pneumonia. The patient seemed to improve up to a week ago, when the temperature and cough persisted, the dulness behind became more marked and spread toward the base of the lung; fremitus absent; voice and breathing bronchial. Needle introduced and pus withdrawn from left side, greenish-yellow, adhesive; heart not displaced; operation advised.

August 1. Child had escaped from observation, and having refused operation passed under care of others. It was brought to me, August 1st, for diarrhœal trouble; chest showed no signs of old trouble; lung had expanded; voice and breathing good, no friction sounds. There was a slight dulness only left over the affected side; fremitus good; no fever; child looks much improved. Mother says that after leaving my care the patient grew worse until she began to cough up large amounts of yellow matter (pus?). This continued some time at intervals; patient recovered; no cough.

Bacterioscopic examination of pus revealed diplococcus pneumoniæ.

CASE XIV.—*May* 13, 1890. L. H., aged eighteen months, male; previous history negative; father and mother well and in apparent good health. Nine days ago child developed a cough, fever and dyspnœa; the dyspnœa increased, fever continued, and there was restlessness at night. When brought to me the child was anæmic; had signs of rhachitis; there was much dyspnœa and moaning respiration, drowsiness, and partial stupor. Temperature 105°; pulse 165; respirations 56. Physical signs of fluid in right side below mid-scapula, needle introduced and a clear bloody serum obtained.

14*th*. Needle introduced, signs of fluid having increased, and withdrew milky pus.

15*th*. Exsection of rib, eight ounces of pus removed.

Bacterioscopic examination showed staphylococcus pyogenes aureus.

CASE XV.—D. H., male, aged five and a half years. Father and mother living, has had no special illness; present illness dates from October 6th, when there was fever and pain and dyspnœa; the pain was referred to the stomach; there was no distinct history of chill. When first seen, October 7th, patient was a well-developed child; no signs of past rhachitis; there was dyspnœa, a dry cough, fever; there was also some restlessness; complains of stomach pain. Temperature 103°; pulse 130; respirations increased. Signs in chest negative.

October 8. Temperature 104°; pulse 140; respirations rapid; pain in right side; rusty sputum. Diagnosis at this time: right lobar pneumonia lower lobe. At end of five days signs pointed to a resolution of an ordinary lobar pneumonia; temperature 102°.

19*th*. Signs of fluid in chest. The boy has not been doing well, and new signs appeared; loss of fremitus; flatness, bulging of right side; subcrepitant râles close to the ear, voice and breathing though coarse heard over whole chest; dyspnœa great; temperature 103½°; pulse 160; respirations rapid.

21*st*. Operation: simple opening in chest and tube inserted; about half a pint of pus evacuated; not fœtid; yellow color.

27*th*. Discharge fœtid and penetrating odor; the drainage bad and a resection was performed, November 5th. Final complete recovery.

Bacterioscopic examination of pus first taken from chest showed diplococcus pneumoniæ.

CLINICAL CLASSIFICATION.

The cases here recorded, from a clinical and bacteriological standpoint, divide themselves quite readily into groups.

GROUP I.—Here we can place those cases of empyema in which the bacterioscopic examination revealed the staphylococcus pyogenes aureus, or the streptococcus pyogenes. These microörganisms did not exist associated, but in so-called pure form and isolated in the exudate. The etiology of these cases is very difficult to make out, because this group does not include cases of empyema where an extraneous source of infection exists. Those cases of empyema in which the pleuritic exudate shows simply the presence of the staphylococcus or streptococcus alone, and where there is no history of traumatism with perforation of the skin, or where there is no suppurating focus of infection outside the pleural cavity, are most puzzling to explain because the bacterioscopic finding gives us no clue as to the origin of the disease. The staphylococcus and streptococci are microörganisms which exist in suppurating processes of the most diverse nature and situation in the body. I have four such cases to place under this heading. One female, aged seven years, and three male children, aged eighteen months, two years, and twelve years respectively. In none of these cases was there a history of traumatism of any kind. When they came under observation, they had been ill with symptoms of pulmonary trouble for periods varying from one to six weeks.

Assuming that an infection of the pleura is most apt to result from adjacent inflammatory processes, the most usual disease preceding or complicating such a pleurisy is pneumonia. Weichselbaum has demonstrated that in addition to the pneumococcus of Fränkel, there exists in the lungs of pneumonic patients the streptococcus pneumoniæ (or pyogenes) and the staphylococcus pyogenes aureus. Though we cannot as yet yield to these microörganisms the dignified position of being the direct cause of pneumonia, we find that they exist in the lung constantly as so-called mixed infections, as they do in diphtheria. Thu[1] has argued that microörganisms in pneumonic fibrinous pleuritis and pericarditis (pneumococci) first find their way through the lymph channels into the subpleural tissue and then into the pulmonary pleura, and finally gain the surface of the pulmonary and costal pleura; from thence they may be carried into the mediastinal spaces and reach the tissue of the pericardium and even the muscular tissue overlying the costal pleura. It is true that the failure to find the pneumococcus in the exudate does not disprove the possibility that at some early stage of the empyema it might have been present; however the tendency to-day is to consider the active predisposing causes exposure to cold and moisture. It is thought that though the above microörganisms may exist in the air passages, as has been proven in the nose and mouth of a healthy individual, they may remain inert until, cold and wet reducing the resistant vitality of the organism, they cause an empyema; so a wound in the chest wall without perforation of the skin may exert a similar influence.

GROUP II.—The cases of empyema which fall under this group are the most interesting of all pleurisies. They are those which either complicate or follow a pneumonia. In the pleuritic exudate of such an empyema the diplococcus pneumoniæ (Fränkel and Weichselbaum) may be demonstrated as in all of my cases.

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[1] Centralbl. f. bakteriol., 1889, Bd. v.

These were two female children, aged thirteen months and two and a quarter years respectively, and seven male children, aged twelve months, two years, two and a half years (two), three and a half years, four years, and five and a half years. They had been ill when they came under observation for periods ranging from one to nine weeks. In one case the patient had been under the observation, from the very beginning, of a skilful observer (Case XV.), and the diagnosis of lobar pneumonia followed by a pleurisy (empyema) was distinctly traced. As recorded elsewhere, the diplococcus pneumoniæ (Fränkel) could be easily recognized in the crude pus by spreading upon cover-glass; beautiful capsule appearances were also obtained. Pure cultures were isolated in every case and their virulence tested upon animals while the cultures were still recent. The experiments were in the direction of intra-pleural or pulmonary injections, so that appearances in the animal experiments resembled very much what is seen in the human subject. If such a pathogenic microörganism is found in a pleuritic exudate, it can be traced to only one probable source, the lung. It is scarcely necessary in any of these cases to prove clinically the existence of a pneumonia. If one will refer to the histories of the cases he will see that in some a pneumonia, either of the lobar or lobular type, must have been in active progress when the empyema was operated upon. At least the physical signs and symptoms indicated this condition of affairs in the lungs upon the opposite side to the empyema, if not in the lung corresponding to the empyema.

In one case, where the patient succumbed, the opposite lung was undoubtedly affected. In other cases, after operation, the temperature would not only persist, but persistent broncho-pneumonic processes were present; this was notably the history of Case III., who continued in the hospital for months, being finally discharged cured. In these cases, tuberculosis was excluded by all possible methods, both clinical and bacteriological. But in the majority of the cases in which the pneumococcus was found in the pus of the empyema, the evacuation of the pus marked the abatement of all symptoms and recovery of the patient, showing that the pneumonia, if it existed, had preceded the accumulation in the chest and had passed through its various stages. The pneumococcus has been found by Weichselbaum in lungs the seat of lobular pneumonia, as well as in those the seat of lobar process.

I have elsewhere described the macroscopic characters of the pleural exudates in the cases of empyema belonging to this group. But in two the pleuritic fluid when first withdrawn from the chest was serous in character, in one case even devoid of flocculi. In both of these cases, the exudate taken later with an exploring syringe was markedly purulent, and both cases were operated upon. In the serous fluids first obtained, as also in the purulent exudate, the diplococcus was found.

This shows distinctly that though an exudate may be serous at first, it may subsequently become markedly purulent, not on account of anything introduced into the chest on the occasion of the first puncture, but from the continued action of microörganisms already present (diplococcus pneumoniæ or streptococcus pyogenes, Fränkel). Again, pus from the chest has a tendency, without this cavity, to separate into strata, the upper one being serous and containing but few leucocytes; we may aspirate this stratum in a marked empyema, and thus can not draw any definite conclusion as to the nature of the exudate. If an exudate contain streptococci, even though serous, we can with certainty predict the advent of pus (Fränkel). We might make a similar assertion of fluids containing the diplococcus pneumoniæ. If a serous fluid withdrawn from the chest fails to reveal any microörganisms upon stain or culture, we can conclude that there is a probable tubercular element in the pleurisy (Fränkel). If such a serous exudate subsequently shows the presence of microörganisms (streptococci) and then becomes purulent, we can suspect contamination. With conscientious cleansing of a needle-syringe, we may fearlessly enter the pleural cavity without having in the least compromised the health of the patient. I am certain much misunderstanding has arisen on this subject through a misinterpretation of the real nature of some of these serous exudates. While some are devoid of microörganisms (tubercular), others are filled with pyogenic microbes, capable not only of producing suppuration, but maintaining it.

GROUP III.—This group, which includes empyema of tubercular nature, is most unsatisfactory in its various aspects. Fränkel, who spent much time and patience upon tubercular pleurisies in the adult, found cases in which examination of the exudate yielded a negative result. So common was this, that he concluded that a negative finding was much in favor of the tubercular character of the exudate. These exudates may be localized or involve the whole pleura. They may or may not be accompanied by lung areas of tuberculosis; in cases where the lung is involved, in the adult, the tubercle bacilli may be found in the sputum, but in children the examination of the sputum is not always practicable. If, as in children, the lung shows no positive involvement and the examination of the exudate is negative, diagnosis and prognosis are difficult. Ehrlich (*Charité Annales*, 1888) explains the absence of tubercle bacilli in two ways: 1. The fibrin formations in the exudate remove the bacilli by enclosing them. 2. Thickening of the pleura by adhesions causes a resistance to the transmigration of bacilli. In empyema, the bacilli are more numerous than in simple pleuritic exudates, because cells passing in myriads into the exudate are more apt to carry bacilli with them. Israel and Gerhart think that the bacilli rather become entrapped in the miliary growths of the pleura, and thus do not pass into the exudate.

I have but one case to place in this group, that of Case II., a boy of eight years, who had had several bronchitic attacks years before the advent of his empyema. The exudate had to be repeatedly examined in order to establish the presence of tubercle bacilli (stained by Ehrlich's method), but the experiments with animals were of a negative character. In this case the streptococcus pyogenes was also found in the exudate from the very first; this is similar to a case in the adult recorded by Fränkel. The formation of adhesions tying down the lung must have been exceedingly great, for operation found the lung unable to expand and repeated exsection of the rib had to be resorted to, in order to obtain partial closure of an immense suppurating cavity. The deformity resulting was most pronounced. I have made attempts in other children and this boy to obtain sputa for examination, but have not succeeded. I attributed my ill success in inoculation upon animals to the presence of contamination in the pus.

Thus, my group of tubercular empyema remains unsatisfactory, though diagnosticated with a certainty from the presence of tubercle bacilli in the exudate. The lungs upon examination yielded no evidences of involvement.

Group IV.—In this group we might class those cases of empyema in which some focus of suppuration situated in another part of the body may be with great probability pointed to as a source of infection. The infecting focus may be adjacent, as in the cases of Fränkel, where a retro-pharyngeal abscess or perforating peritonitis were the cause of the empyema, or the empyema may be one of the manifestations of a species of pyæmia (as in my own case). This was a male infant, aged four months, who was perfectly well up to within two weeks of its death. The infant was vaccinated and then developed a burrowing abscess of the foot, and after this the empyema appeared. No autopsy was allowed; it would have been interesting to see (though no pneumococcus was found in the exudate of the pleura) whether a pneumonia (pyæmic) was present with the empyema. The rapid death of the child speaks in favor of some virulent infection; an autopsy might have revealed several other hidden foci of suppuration.

The bacterioscopic finding and experiments in the above case are interesting. The streptococcus which was isolated and existed alone in the exudate was certainly very virulent. Two animals injected with a pure culture (15 and 16), both died. Both were injected at the same time with the same culture; the one in the pleural cavity, in this case, the animal died in two days; streptococci were found in the blood and organs, but there were no inflammatory exudates; the kidneys were swollen and the spleen was enormous in size. The second animal was injected subcutaneously, and the report of the autopsy shows metastatic abscesses all over the body with general jaundice and enlarged spleen and cloudy kidneys. These results differ widely from those with streptococci obtained from cases of Group I. Here the animals survived and autopsies after months showed no effects of infection, or as in rabbit 14, only the effects of local processes limited in nature.

PUTRID EMPYEMA.

By this I desire to designate those cases of empyema in which there was a putrid or fetid odor to the discharge, and in which this odor persisted.

In both of my cases the exudate turned putrid after operation. In one (XV.) the empyema followed a pneumonia, and in the fluid first withdrawn from the chest, the pneumococcus alone existed. In this case, on account of the imperfection of drainage due to the first mode of operating, there was retention of pus, the chest wound finally closed, and when reopened again for resection, the pus was very putrid.

The second case was that in which the pus showed the presence of tubercle bacilli. There was no odor to the pus upon the first resection. But there was not only lack of any expansion in the lung, leaving an immense cavity in the chest, but the pus remained in this cavity. Here the empyema became putrid, necessitating repeated washings of the chest with creolin solution. In spite of this fact, the pus remained putrid. Investigation succeeded in revealing the presence of a fluorescent green bacillus, rather thick and short, which fluidified gelatin (much like that found in water) and grew upon agar of Weichselbaum in a dark-green layer with a rather mawkish odor to the growth. This is all that could be found in addition to the tubercle bacillus and streptococcus previously present. This shows that the most innocent exudate may turn putrid, as in the case of pneumonic empyema, as well as the tubercular cases. It seems rational to suppose that lack of expansion of the lung or retention of pus, especially where the chest has been injected with so-called antiseptic solutions (in the above cases boric acid or creolin), are conditions peculiarly favorable to the development of putrid exudates. I have seen air enter daily freely into the chest cavity without causing the pus to become putrid; also the introduction of bacteria from the lips of the wound was unavoidable, on account of the free moving of drainage-tubes and dressings and tampons over the mouth of the external wound. Yet drainage being free, with full lung expansion, no putridity appeared. In the future, the appearance of a putrid exudate in a chest in which primarily the pus possessed no such characters, would make me think of retention due either to insufficient drainage or want of proper expansion of the lung.

PROGNOSIS.—We can safely say that the present status of our knowledge in these cases enables us to assure our patients with moderate certainty as to the outcome of the illness of their children. Such prognosis may be made at the bedside or within a very short period from the first examination of a case. The simple means at the disposal of every clinician are adequate for the examination of crude specimens of pus, and delay of a day or two at the most is all that may be necessary for more deliberate investigation. Nor do I think that we should take too much credit to ourselves for the recovery of a certain class of these cases which, under proper guidance, invariably get well. Thus, it would appear that the best prognosis is held out to those which belong to Groups I. and II. More especially is this true of cases of meta-pneumonic empyema. In one of these (XIII.), in which the purulent exudate filled the chest and was withdrawn from the same by exploring needle and contained pneumococci, the patient recovered; having refused operation, the empyema perforated the lung and thus gained exit externally, pus being coughed up. Subsequent examination of this case revealed nothing abnormal on the diseased side but slight dulness. The lung had thoroughly expanded. Fränkel records similar cases in the adult. It would scarcely be well to expect too much from such *laissez faire* treatment, for a large percentage (Ziemssen's *Vorträge*) of cases which are allowed to perforate die. The age of the patient and the presence or absence of any considerable pneumonia will weigh in each case. Some authors are enthusiasts for the simple opening and drain in the chest; others insist on the trial of repeated aspiration; others contend that resection of the rib is the only safety of the patient and secures a rapid expansion of the lung. The truth lies in the etiology of the cases of empyema. In the meta-pneumonic exudates cases will do equally well by the various modes of treatment; in other words, the pus evacuated by whatever means, there is an inherent tendency to absorption of what inflammatory product there is remaining and return to a normal status, as in the pneumonic lung.

The pneumonic exudates, if they hold out such brilliant prospect of recovery to the patient, are a striking contrast to the tubercular cases, where only a partial recovery is at best possible. There are cases on record where, especially in children, an empyema (Ziemssen's *Vorträge*) of established tubercular nature has made an apparently good recovery, yet we must doubt its permanency. We cannot rid ourselves of the idea that a large extent of surface like the pleura, once tubercular, must, if recovery takes place, be a latent danger to the patient.[1] In other cases, the recovery results with fistulæ, retraction, and all the concomitants of permanently crippled respiratory apparatus. The pyæmic cases are fatal as far as we know.[2]

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[1] Klesch and Vaillard (Arch. de Physiol. et Path., 1886) have made autopsies upon pleurisies of a tubercular character which had apparently recovered, and were able to prove their tubercular nature at autopsy.

[2] I desire to express my sincere obligations to Dr. Barium Scharlau, of the Mount Sinai Hospital, for the clinical material which he so kindly placed at my disposal.